ADVANCED ASSESSMENT
Shock

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References – Emergency Medicine

Shock

Widespread Tissue Hypoperfusion

Results In

Inadequate oxygenation and supply of nutrients at a cellular level
Inadequate removal of metabolic waste products

Due To

Inadequate circulation of blood

Leads To

Generalized impairment of cellular function
Ultimate cell death

ENERGY PRODUCTION

Human Body Likes Presence of Oxygen
To Produce Energy

Aerobic Metabolism

- Energy production in the setting of oxygen
- High amount of energy production
  - 38 Adenosine Tri-phosphate
  - Low waste (Acid Production)

Anaerobic Metabolism

- Energy production without oxygen
- Low amount of energy production
- 2 Adenosine Tri-phosphate (Glycolysis Sugar Splitting)
- High acid production
- Pyruvic to Lactic Acid results in metabolic acidosis
- Cell functions cease due to lack of energy and acidosis
- Cell death

S Septic, Spinal

H Hypovolemic

O Obstructive, Mechanical

C Cardiogenic

K Anaphylactic

Septic Shock

- Caused by serious systemic bacterial infection (usually gram negative bacteria)
- Causes a release of vasoactive agents that affect microcirculation (arterioles and venules dilate {increased container size}, capillary leak {loss of volume}
- Increased container size and loss of volume lead to inadequate tissue perfusion

Septic Shock

- **Signs and Symptoms**
  - Fever (may not have fever), tachycardia, tachypnea, confusion, petechiae around eyes, purpura
  - Look for history of recent infection (bladder, pneumonia)
- **Treatment**
  - Identify and correct infection (antibiotics)
  - Manage hypovolemia and acid base imbalance (fluid therapy, vasoactive and cardiac drugs)

Neurogenic Shock/Spinal Shock

- Predominantly causes by Spinal Cord Injury, may also be caused by head injury, CVA or drug overdose
- Block of sympathetic outflow results in peripheral vasodilatation

Spinal Shock

- Vasomotor paralysis below level of spinal cord injury
- Normal vasomotor tone through sympathetic control is lost
- Results in vasodilatation, increased container size
- Even normal intravascular volume is insufficient to fill vascular compartment
- Unable to mount compensatory tachycardia
- Heart rate remains unchanged with hypotension
- Look for line where sympathetic response ends

- **Treatment**
  - Similar to Hypovolemia, caution not to cause fluid overload

Mechanical Shock/Obstructive

- Blood flow disrupted due to mechanical obstruction
- Examples
  - **Tension Pneumothorax** - Increased Intrathoracic pressure impedes low pressured venous return, increased pressure can twist and compress heart
  - **Cardiac Tamponade** - Compression of heart due to accumulation of fluid or in pericardial sac (usually due to trauma or infection)

Mechanical Shock/Obstructive

- Examples cont'd
  - **Pulmonary Embolism** - Blockage of blood flow in pulmonary circulation (fat, air, thrombus)
  - **Aortic Dissection** - Blood flow obstructed distal to left ventricle (Dissection of Aortic Arch= b/p right arm greater than left)

- **Treatment**
  - Correct cause of obstruction

Cardiogenic Shock

- Cardiac action cannot deliver circulating blood volume adequate for tissue perfusion
- **Examples**
  - Massive M.I
  - Rhythm Disturbance (fast or slow)
  - Cardiac Contusion
- **Treatment**
  - Improving pumping action of the heart and managing cardiac rhythm irregularities (medications, electrical therapy, fluid)
  - Treat associated problems ex. CHF

Cardiogenic Shock - VISCIOUS CYCLE

- Heart Damage >50% Muscle Damage

→ Decreased Circulating Volume
→ Increased Compensatory Heart Rate
→ Damaged ISCHEMIC Heart Has Increased Workload, Increased Oxygen Demand

→ FURTHUR Damage To Heart Muscle
→ B/p Doesn't Change Or Drops
→ CON'T Cycle Till Complete Failure
→ >90% Mortality Rate

Anaphylactic Shock

- Severe systemic allergic reaction due exposure to an allergen
- (ex. food,drugs,insect bites)
- Causes exaggerated release of chemical mediators (HISTAMINE, serotonin, kinins)

Multi-System Involvement

- Respiratory
  - Upper airway obstruction secondary to edema
  - Severe bronchiole constriction
- Integumentary
  - Hives
  - Local Swelling (capillary leak)
- Gastrointestinal
  - Nausea and vomiting, Abdominal pain, Diarrhea

Multi-System Involvement

- Circulatory
  - Hypotension (Capillary leak, vessel dilation)
- Treatment
  - remove exposure of allergen, epinephrine, bronchodilators, antihistamines, fluid therapy

Hypovolemic Shock (Loss of Circulating Volume)

- Hemorrhagic
  - Internal blood loss, eg. G.I bleed
  - External blood loss, laceration
- Non-Hemorrhagic
  - G.I losses prolonged severe vomiting and diarrhea
  - Renal losses excessive use of diuretics (^urine prod.)
  - Cutaneous losses heat exhaustion and burns (3rd spaced shift)

TREATMENT

Correct circulatory deficit and cause, surgery, volume replacement

Normal Circulating Volumes

Adult = 70 ml/kg
Pediatric = 80 ml/kg
Infant = 90 ml/kg

Stages of Shock

1st Stage: 0-15 % of blood volume

2nd Stage: 15-30% of blood volume

3rd Stage: 30-40% of blood volume

4th Stage: >40%

Early Signs & Symptoms of Shock

- Altered LOA, confusion, agitation
- Normal possible slight increase in blood pressure
- Tachycardia
- Pale, moist skin

Late Sign

- Hypotension

3 STAGES OF SHOCK

1 Compensated Shock

- Some decreased tissue perfusion, but bodies compensatory mechanisms overcome
- Cardiac output and systolic b/p maintained by catecholamine release
- Mild tachycardia, altered LOA, agitation, delayed cap refill, cool skin, b/p normal or slightly elevated
- Continued compensatory leads to acidosis as perfusion decreases (chemoreceptors respond with increased respiratory rate)

2 Uncompensated Shock

- Unable to maintain systolic blood pressure
- Systolic and diastolic blood pressure drops, pulse pressure narrows till non existent (pulse pressure systolic minus diastolic)
- Compensatory mechanisms begin to fail, cerebral blood flow decreases even with blood being shunted to vital organs
- Moderate tachycardia, confusion → unconsciousness
- Delayed cap refill, cold extremities, cyanosis, hypotension

3 Irreversible Shock

- Progression of cellular ischemia and necrosis with subsequent organ death, despite restoration of oxygenation and perfusion
- Membrane pump fails and various organelles breakdown inside of cell
- Bradycardia, dysrhythmia, coma, Frank Hypotension
- Results in death even if volume corrected

COMPENSATORY MECHANISMS IN SHOCK

Neurogenic Compensation (Central Nervous System)

- Autonomic (Sympathetic, Fight or Flight)
- Rapid onset
- Increased heart rate (chronotropic), increased force of contraction (inotropic), increased conduction (dromotropic) Beta 1
- Stimulates Adrenal Medulla to secrete Norepinepherine and Epinephrine
- Nor Epinephrine-Alpha 1 peripheral vasoconstriction, Epinephrine- Beta 2 dilate vessels to coronary and skeletal muscle, bronchodilation

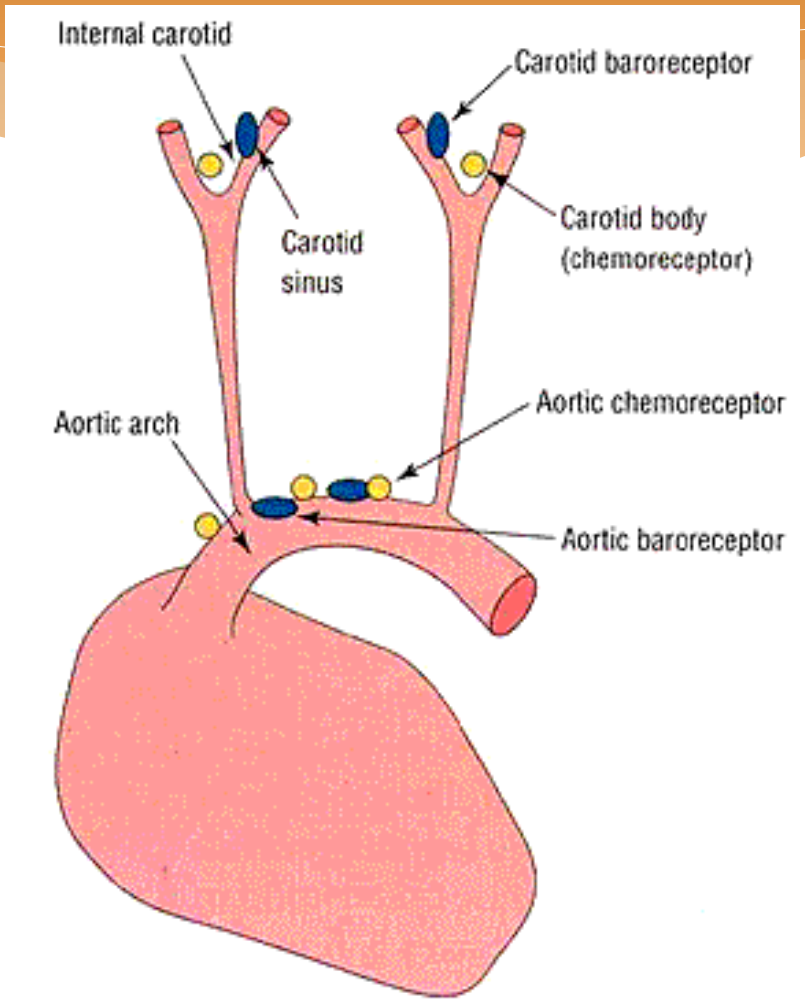
Baroreceptors and the Sympathetic Nervous System

- Controls rapid moment to moment B/P
- ⇩ in blood pressure causes baroreceptors (stretch) to send fewer impulses to the cardiovascular center in the CNS
- Response ⇧ Sympathetic output

  ⇧Cardiac Output

  Vasoconstriction

  =⇧ in blood pressure



Chemical Compensation (Respiratory System)

- Decreased Po2 sensed by peripheral chemoreceptors, increased acidosis sensed by central chemoreceptors
- Stimulates increased respiratory rate, maximizes FiO2, compensates for acid base imbalance

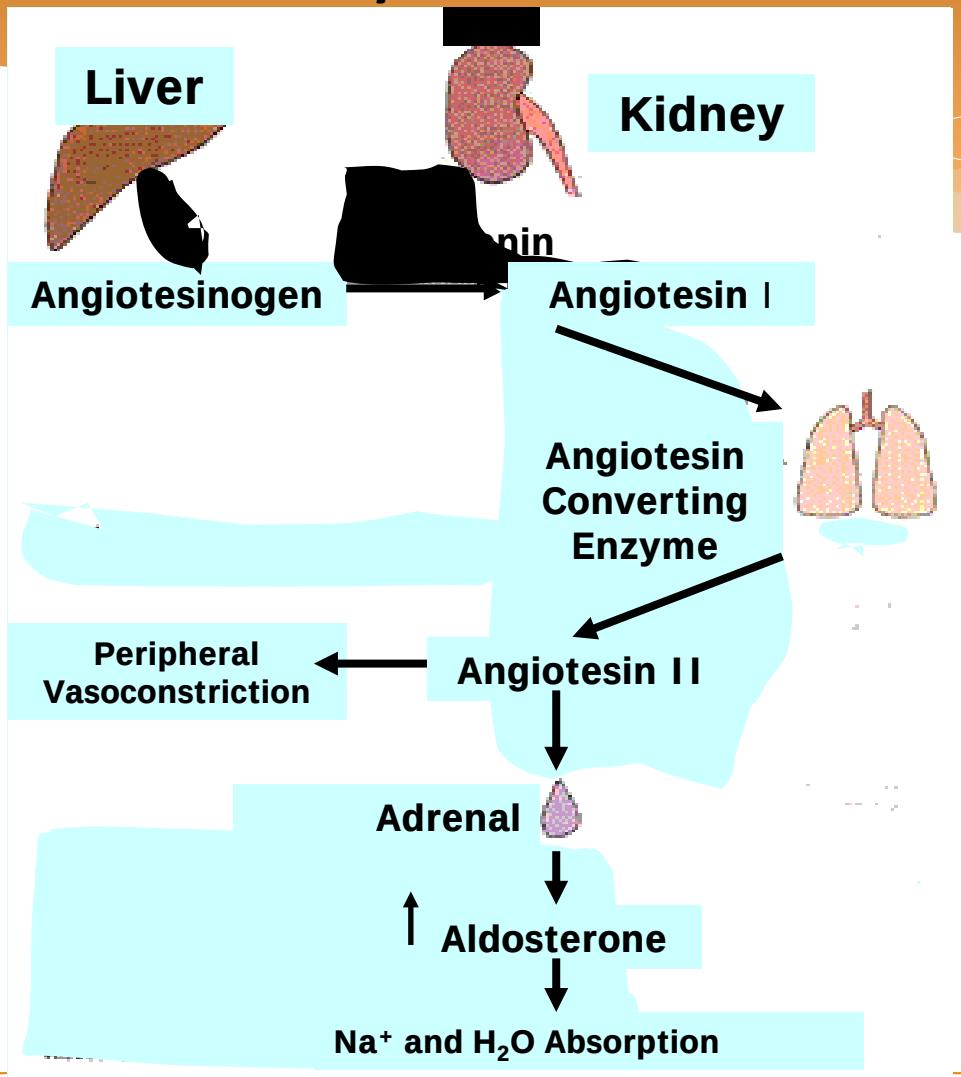
Hormonal Compensation

- Epinephrine and Nor Epinephrine as above
- Renin-Angiotensin System
- Anti-diuretic Hormone
- ACTH – Adrenocorticotropic Hormone

Renin-Angiotensin System

- Decreased blood flow to Kidneys-> Hypoprofusion sensed by Juxtaglomerular Cells, causes kidneys secrete renin->Renin acts on Angiotensinogen (protein from liver)->Angiotensine 1->at lungs converted to Angiotensine 2 by ACE-> Angiotensine 2 powerful vasoconstrictor and stimulates Adrenal Cortex to secrete Aldosterone
- Aldosterone stimulates kidneys to retain sodium with that water overall increasing fluid volume

Renin/Angiotensin System



Anti-diuretic Hormone - ADH

- Secreted by posterior pituitary if stimulated by hypothalamus (Senses increased osmotic pressure)
- ⇩volume = ⇧ Tonicity (sodium)
- Causes tubules of kidney to be more permeable to water therefore more volume retained

ACTH - Adrenocorticotropic Hormone

- Hypothalamus stimulates anterior pituitary to secrete ACTH
- ACTH stimulates metabolism of carbohydrates, proteins and fats
- Decreases permeability of capillary walls helping prevent loss of intravascular fluid

SPECIAL ASSESMENT CASES

Relative Hypotension

- Hypotension is usually defined as blood pressure less than 90 or 100 mmhg (average person)
- Patients who have Hypertension will have decompensatory drop in blood pressure, their drop of 30 or 40 mmHg may only put them in the 110,100 systolic range they are just as hypotensive though as the person normal tensive with blood pressure of 80mmHg
- This is called relative hypotension

Beware of Beta Blocked Patient

- Will not have compensatory increase in heart rate

Beware of the Pediatric Patient

- Compensate until point of failure
- Shock may be present despite normal blood pressure
- Look For:
- Altered LOA, diminished peripheral pulses, delayed cap refill, Tachycardia, check temperature of extremities
- Late signs: Bradycardia, hypotension

Shock in Pregnant Female

- 30-40% blood volume increase
- Only 15-20% increase in hematocrit (functionally anemic)
- Present in shock like picture without deficit {Functional Hyperventilation, $PaCO_2$ 30mmhg, Lower b/p, Increased heart rate 10-30bpm
- Will appear in class 2 or 3 shock without injury

Burn Patient

- Burn patient with 2nd and 3rd degree burns have large amount of fluid loss due to third space shift
- Damage causes fluid to shift from intracellular space to interstitial space
- Leads to hypotension and large amounts of swelling in burned area

- Parkland Formula
- Ideal fluid replacement (4ml/kg X % of area burned)
- 1st half over 8 hours, 2nd half over next 16 hours

Orthostatic Vitals

- Body unable to compensate for drop in blood pressure while in sitting or standing position
- Lying flat allows body to maintain somewhat normal blood pressure
- Sitting patient up will cause change in blood pressure and heart rate
- Drop of 20mmHg systolic or 10 mmHg diastolic is significant
- Increase of heart rate 20 per minute is significant
- Patient must be in sitting position for 1-2 min
- Do not do on patients exhibiting signs of hypoperfusion (dizziness lying flat)
- Done to rule out positional change in vitals

Well Done!

Ontario Base Hospital Group

Self-directed Education Program